healed and the inflammation has left the skin. Their attention has been so thoroughly centered on the swelling and pustulation of the disease that they have given no consideration to the fact that scars are being formed. These scars become noticeable after the swelling has gone. The physician and the x-ray treatment are blamed for this unfortunate condition.

Dr. Henry D. Niles of New York has published an article in the January, 1933, Archives of Dermatology and Syphilology in which he gives the record of forty cases which he treated in a manner to compare the effect of x-ray in the production of scarring with other methods of treatment of various types of acne. The summary of this article is as follows:

1. In forty patients who were given an average of twelve and one-half weekly exposures of one-fourth skin unit of roentgen rays on one side of the face, and a placebo treatment on the other, the scars were equal on both sides in thirty-two cases, more pronounced on the untreated side in five, and greater on the treated side in three.

2. The amount of scarring after acne vulgaris depends on the severity and duration of the eruption and the tendency of the patient's skin toward scar forma-tion. This seems to be neither increased nor decreased by roentgen therapy.

3. In nineteen of the forty patients, the untreated side was either entirely cured or almost well, and as much improved as the treated side. Several theories as to the possible explanation of this unexpected finding are given.

The above series naturally will have to be added to considerably before we can make a proper statistical survey; however, they do definitely prove that we are certainly not damaging our patients through the use of proper x-ray therapy, and that it is probably preferable to other types of treatment in cases in which the damage to the skin is relatively severe.

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SALT SUBSTITUTION THERAPY IN ADDISON'S DISEASE

It has been demonstrated experimentally that a loss of sodium and chlorin particularly, as well as a negative balance for calcium, magnesium, potassium, phosphorus and nitrogen occurs in adrenalectomized animals.1 Because of the loss of these basic elements, Rubin and Krick directed their treatment toward replacing them; and the improvement which followed was striking: untreated controls died in ten days, whereas rats receiving the salt mixture survived in apparent health for four months. These investigators believe it is probable that the action of the adrenal cortical hormone is primarily one of salt regulation. It is known, further, that sodium excretion is augmented in adrenalectomized animals.2

Loeb and co-workers 2 have observed a decrease in the sodium concentration of the blood serum in Addison's disease patients, and by diminishing the salt intake they have promptly induced symptoms of adrenal insufficiency. Administration of large amounts of sodium chlorid, on the other hand, brings about marked clinical improvement, which parallels the sodium level of the blood serum. Harrop et al.,3 and Rogoff 4 appreciate this also, since Harrop suggested the use of a saltfree diet as a diagnostic measure. Rogoff has noted a "spectacular resuscitation from coma by intravenous saline solutions."

Our experience in maintaining an Addison's disease patient in apparent health, with sodium chlorid substitution therapy alone, confirms these observations. In this instance, the cost of cortical extract was excessive for this individual, so salt was prescribed. Eschatin had been given in recommended amounts over a four weeks' period, with some improvement, but the patient was not able to leave her bed. A salt-poor diet for a tenday period aggravated the typical symptoms of Addison's disease. Blood (plasma) sodium chlorid was 508 milligram per cent before treatment (normal range 570 to 620 milligram per cent). Ten grams of table salt were given orally daily and eschatin was discontinued. Within ten days the patient was up daily, and in two weeks after onset of therapy she was able to care for herself entirely. She has since been well, except for ankle swelling which occurs when her blood salt level exceeds the normal range. Over a four months' period the blood (plasma) sodium chlorid content has ranged from 564 to 653 milligram per cent.

In our opinion, salt substitution therapy should be used in maintaining the normal level of sodium chlorid, as well as other elements found to be deficient in these individuals. It is probable also that the administration of sodium chlorid exerts some sparing action on other mineral metabolism. Since but approximately 10 per cent of the normal amount of adrenal cortical tissue is needed for life, and since glandular therapy is expensive and not entirely satisfactory when used alone, more attention should be paid to salt metabolism in Addison's disease. Increases in blood non-protein and urea nitrogen have been taken to indicate abnormal trends in adrenalectomized dogs.5 These tests might be used clinically, together with sodium chlorid determinations as guides for ascertaining whether salt substitution or glandular therapy is indicated.*

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^{*} With technical assistance of Miss T. Blumberg.